To submit questions for this regular feature, please send them to the Journal of Psychiatry & Neuroscience / Revue de psychiatrie & de neuroscience, *Canadian Medical Association, 1867 Alta Vista Dr., Ottawa ON K1G 3Y6, Canada; fax 613 729-9545; jpn.office@sympatico.ca Please include details of any relevant case and your name, address, telephone and fax numbers as well as your email address.*

How should lithium-induced thyroid dysfunction be managed in patients with bipolar disorder?

Lithium, still a major treatment for bipolar affective disorder, is known to have thyrostatic effects; the clinical implications of this are not completely understood, however.

Mr. B, a 42-year-old man, presented with an initial episode of mania. After an appropriate workup and once he was behaviourally stabilized, lithium was introduced. Routine screening 6 weeks after treatment was initiated showed an elevated thyrotropin (TSH) level of 7.6 mU/L and normal thyroxine (T4) and triiodothyronine (T3) levels. The patient remains stabilized, with no evidence of depression or mania.

Lithium inhibits thyroid function at various points in the thyroid axis. A large number of studies have shown that anywhere from 0% to 47% of patients on long-term lithium treatment will develop clinical hypothyroidism. Cases of subclinical hypothyroidism are also frequent, particularly in female patients and those with rapid cycling disorder.

A major issue remains as to when clinical intervention is required. Earlier studies showed that transient elevations of TSH could occur with lithium therapy for bipolar disorder, particularly during the initial 2–3 months of treatment. As long as the elevations are modest, no intervention is required, although continued monitoring of thyroid function is indicated. No evidence of mood disturbance is further justification for no specific intervention.

There are 2 situations in which intervention may be required. The first is when a patient has refractory mood disorder, particularly rapid cycling, with modest elevations of TSH (i.e., subclinical hypothyroidism). Here, thyroid supplementation with T4 may be indicated to remove one of the predisposing factors to mood instability and treatment refractoriness. Interestingly, there is a limited literature documenting the efficacy of high-dose T4 as a mood stabilizer, irrespective of baseline thyroid status, and especially when it is used in conjunction with other mood stabilizers (see Bauer MS, Whybrow PC. *Arch Gen Psychiatry* 1990;47:435-40). The second instance would be if there is evidence of clinical hypothyroidism, and thyroid hormone is required as replacement therapy. It is important to note that discontinuation of lithium is not required when lithium-induced subclinical or clinical hypothyroidism supervenes. Rather, thyroid replacement therapy should be used when indicated, and the bipolar disorder should be managed in the most appropriate way. In other words, if lithium is still indicated for the patient, it should be continued along with thyroid replacement therapy. The use of alternative mood stabilizers to reverse the lithium-induced thyroid dysfunction is not the optimal management of the bipolar patient.

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The information in this column is not intended as a definitive treatment strategy but as a suggested approach for clinicians treating patients with similar histories. Individual cases may vary and should be evaluated carefully before treatment is provided.

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